Chapter 6: Radiation Risk Assessment

6.1 Introduction

The U.S. Environmental Protection Agency (EPA) recommends that both a chemical risk assessor and a radiation risk assessor be on site for a Remedial Investigation/Feasibility Study. This is because there are major differences in the procedures used to characterize chemical and radionuclide contaminants. This chapter defines the differences between chemical and radiation risk assessments.

The principal adverse biological effects associated with ionizing radiation from radioactive substances in the environment are carcinogenicity, teratogenicity, and mutagenicity. Carcinogenicity is the ability to produce neoplastic changes that result in carcinomas or cancerous tumors. This is a stochastic effect and is considered to be the limiting deleterious effect at the dose levels expected at DOE Superfund sites. Teratogenicity and mutagenicity are the nonstochastic, noncarcinogenic effects associated with exposure to radiation. Teratogenicity is the ability to induce or increase the incidence of congenital malformations, which are permanent structural or functional deviations produced during embryonic growth and development. Mutagenicity is the ability to induce genetic mutation in the nuclei of either body cells or reproductive cells.

Radiation-induced genetic effects have not been observed in human populations, and extrapolation from animal data reveals that risks per unit exposure are smaller than, or comparable to, the risk of cancer. In addition, the genetic risks are spread over several generations. ‘he risks per unit exposure of serious teratogenic effects are greater than the risks of cancer. However, there is a possibility of a threshold, and the exposures must occur over a specific period of time during gestation to cause the effect. (Teratogenic effects can be induced only during the nine months of pregnancy, whereas genetic effects are induced during the 30-year reproductive generation and cancer can be induced at any point during the lifetime.) Therefore, the cumulative risk of cancer maybe many times greater than the risk of genetic or teratogenic effects due to the potentially longer period of exposure. Consequently, the carcinogenic effects typically are used as the sole basis for assessing radiation-related human health risks of a site contaminated with radionuclide.

The organization of this chapter is slightly different than that of the preceding chapters. The preceding chapters discussed one aspect of the baseline risk assessment, while this chapter discusses all aspects of the risk assessment as it is related to radiation. After the discussion of the statutory and regulatory history, the chapter is organized into sections on exposure, toxicity, and risk The conclusion section will summarize the issues that are pertinent to radiation which may be important in a regulatory dialogue.

6.2 Discussion of Radiation Risk Assessment in Statutes and Regulations

6.2.1 Statutory and Regulatory Radiation Risk Assessment Framework

CERCLA was enacted to reduce and mitigate human exposures to release-s of "any pollutant or contaminant which may present an imminent and substantial danger to the public health or welfare" (CERCLA, 1980). The definition of a hazardous substance in Section 101(14) of CERCLA includes "any element, compound, mixture, solution, or substance designated pursuant to section 102 of this Act" and "any hazardous air pollutant listed under section 112 of the Clean Air Act." Section 102(a) of CERCLA states that "the Administrator shall promulgate and revise as may be appropriate, regulations designating as hazardous substances, in addition to those referred to in section 101(14) of this title, such elements, compounds, mixtures, solutions, and substances which, when released into the environment may present substantial danger to the public health or welfare or the environment." In 40 CFR Part 302.4 the Administrator listed radionuclide as being hazardous substances. Section 112(b) of the Clean Air Act lists radionuclide as being hazardous air pollutants. Thus, radionuclide are incorporated into the baseline risk assessment protocol through statutes and through the regulatory process.

6.3 Exposure

6.3.1 Exposure Pathways

Assessors use mathematical extrapolation models (e.g., EPA's computer model "RADRISK") to quantify the relationship between cancer incidence and exposure to radioactive materials. These models estimate the largest possible linear slope (within the 95 percent Upper Confidence Limit) at low extrapolated doses consistent with the data. This "radiocarcinogenicity slope factor" is the "maximum likelihood estimate of the age-averaged lifetime total excess cancer risk per unit intake or exposure" (EPA's IRIS-computer data base). Assessors use the RADRISK computer code to estimate dose rates from ingestion or inhalation of radioactive materials. The DFSOIL code is used to calculate kerma and energy flux of photons in the air at one meter above the ground surface. Kerma, which stands for K inetic E nergy R eleased in M aterial, is defined as a unit of exposure, expressed in rads, that represents the kinetic energy transferred to charged particles per unit mass of irradiated medium when indirectly ionizing (uncharged) particles, such as photons (x-rays or gamma-rays) or neutrons, traverse the medium. (If all of the kinetic energy is absorbed "locally," the kerma is equal to the absorbed dose.) Then the DOSFACTOR code is used to convert the estimates of air kerma obtained from DFSOIL to organ dose rates. To generate the slope factors, assessors use the calculated dose rate with risk models in conjunction with life table analysis. However, the true risk to humans, although not identifiable, is not likely to be the upperbound estimate; it may, in fact, be lower. EPA's Office of Radiation and Indoor Air (ORIA) calculates cancer slope factors for radionuclide of potential concern at Superfund sites. These values are listed in EPA's Health Effects Assessment Summary Tables (USEPA, 1994a), not IRIS.

*Rad stands for Radiation Absorbed Dose and is a measure of the energy imparted to matter by radiation. 1 Rad - 100 ergs per gram.

The pathways of exposure and the mathematical models used to evaluate the potential health risks associated with radionuclide in the environment are similar to those used for evaluating chemicals of concern, except that external radiation is unique for radionuclide and inhalation of volatiles and dermal absorption are more significant exposure pathways for chemicals than for radionuclide. The behavior of a radionuclide in the environment with regard to its transport and inter-media transfer is determined by the same physical/chemical processes that govern chemical contaminants. Consequently, the types of data needed for a radiation risk assessment are similar to three required for a chemical risk assessment. The primary differences lie in the procedures used to characterize the radionuclide contaminants. For example, in addition to exposure due to ingestion, inhalation, and direct contact, radiation emitted from photon sources (i.e., gamma rays) is an external penetrating exposure. Therefore, gamma emitters, whether they are internal or external, are important in risk assessments. Alpha and beta emitters are only important in radiation risk calculations when they are internal (inhaled or ingested).

For inhalation and ingestion, the slope factors are the central estimates (i.e., median or 50th percentile values) of the age-averaged, lifetime excess cancer incidence (fatal and non-fatal cancer) risk per unit of activity intake. For external exposure, the slope factors are the central estimates of the age-averaged lifetime excess cancer incidence risk for each year of exposure to a unit activity concentration of photon-emitting radionuclide. They can also be important in skin absorption depending upon the "carrier" chemical with which the radionuclide is associated (mixed waste) or attached (radio-labelled or "tagged"). If the radio-labelled or mixed waste chemical is absorbed through the skin, then the alpha- or beta-emitting radionuclide would be considered as an internal emitter (e.g., dimethyl sulfoxide [DMSO]) mixed with a radionuclide, or DMSO labelled with Carbon-14). If the alpha- or beta-emitters are not mixed with or tagged to a dermally absorbed chemical, then internal exposures to these radionuclide should not be considered (USEPA, 1994b).

6.3.2 Exposure Assessment

Despite the differences between the way exposures are expressed for radionuclide and chemicals, the approach to exposure assessment is essentially the same, with the following exceptions: the consideration of external (penetrating photon radiation) exposures, conversion of radiation exposures to dose equivalents, and the fate and transport models must be made specific to radiation exposure to account for the ingrowth and decay processes of radionuclide. Additionally, different time scales must be considered in a radiation risk assessment because of the radioactive decay process. For example, some radionuclide not only "decay away" but there is an "ingrowth" of "new" radioactive decay products. For example, plutonium-241, which has a half-life of 14.4 years, decays to americium-241, which has a half - life of 432.2 years. 'his illustrates an example in which the daughter is longer lived and more toxic than the parent. Therefore, each such radioactive progeny may also become dominant contributors to the total radiation exposure assessment over a period of several hundred years.

6.3.3 Radiation Dosimetry

Radiation dosimetry can be defined as the amount of energy deposited in living tissue due to internal and external exposures to ionizing radiation. The potential adverse effects of this amount of energy deposited in living tissue are proportional to energy deposition. Therefore, the term "dose," when

used in radiation exposure, is defined as the energy imparted to a unit mass of tissue, whereas "chemical dose" means the mass of chemical penetrating into an organism.

6.4 Toxicity

6.4.1 Toxicity Assessment

The first step in a toxicity assessment for radionuclides is hazard identification, which is a determination of whether exposure can increase the incidence of an adverse health effect. Then, a dose response assessment is used to quantify the toxicity and characterize the relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population. There is no need for an extensive discussion of toxicity, mainly because exposure to any radioactive substance is, by definition, assumed to be hazardous (USEPA, 1989a). An extensive body of literature exists on radiation carcinogenesis in man and animals that indicates that ionizing radiation can be considered "pancarcinogenic" (i.e., it acts as a complete carcinogen in that it serves as both an initiator and a promotor and can induce cancers in almost any tissue or organ).

Each radionuclide produces unique radiation characteristics that can affect different organs in the exposed individual. **EPA has** calculated the annual radiation dose equivalent from each radionuclide to each organ dose from a continuous lifetime exposure with a constant exposure rate. Using these calculations, assessors can estimate the average excess number of all types of radiation-induced fatal cancers per year for the corresponding dose equivalents received during that year and relevant preceding years. By using epidemiological data, extrapolation from high radiation doses to low doses, and hypothetical models for projecting risk through a lifetime, the excess number of radiation-induced fatal cancers can be determined. EPA has also calculated each radionuclide slope factor by dividing the excess fatal cancer risk for that radionuclide by the mortality-to-incidence risk ratio for the types of cancer induced by that radionuclide (USEPA, 1989b).

This helps assessors evaluate cancer incidence and determine the probability that fatal cancer will occur at a particular site. The use of the mortality-to-incidence risk ratio and the site-specific relative risk model are the major differences between estimating cancer risk for radionuclide and estimating it for chemical carcinogens. This is because EPA does not incorporate the mortality-to-incidence ratio in the cancer slope factors for chemical carcinogens. Chemical carcinogen risks are calculated on cancer incidence, while radionuclide cancer risks are calculated on cancer deaths. (Chemical carcinogens are based on laboratory animal experiments and radiation cancers are based on groups of humans exposed to low-LET radiations, such as the Japanese atomic bomb survivors and medical patients treated with radiation). The mortality-to-incidence ratio is used for radionuclide to convert cancer deaths to cancer incidence. The underlying population rates for mortality and incidence vary differently with respect to age. Therefore, age-specific incidence is greater than mortality, that is, it increases less steeply with age.

Most sources of environmental contamination come from inhalation and ingestion rather than external exposure and result in a nonuniform distribution of radioactive material in the body so that some organ systems receive much higher doses than others. For example, since iodine radioisotopes concentrate preferentially in the thyroid, the dose to this organ can be orders of magnitude larger than the average dose

to the body. To determine the probability that fatal cancer occurs at a particular organ site, EPA has used the incidence risk coefficients and mortality-to-incidence ratios from the BEIR V report (NAS, 1990). However, because not all forms of thyroid cancer can be induced by radiation and, for those that are, a more reasonable mortality-to-incidence ratio would be 0.1, EPA has used that value in its calculations. Likewise, lung cancer incidence and mortality have both shown an increasing trend between 1970 and 1980. Since incidence precedes mortality, an uncorrected mortality-to-incidence ratio gives a low estimate of the fraction of those persons who, having been diagnosed with lung cancer, will die of that disease. Therefore, a mortality-to-incidence ratio of 0.94, based on long-term survival studies by the National Cancer Institute for lung cancer, has been used. It is generally accepted that the risk estimates for the individual sites are less certain than are the risk estimates for all sites combined.

EPA discusses in its recent report, "Estimating Radiogenic Cancer Risks" (USEPA, 1994b) that it has revised its methodology for &riving radionuclide slope factors in the HEAST document- Instead of using the BEIR III dosimetry, EPA used BEIR V dosimetry in the revised methodology. The relative biological effect (RBE) value for alpha particles has been revised to 20 from 8, except for leukemia (RBE is 1) and breast cancer (RBE is 10). For all cancers (except breast cancer), EPA assumed a dose and dose rate effectiveness factor (DDREF) of 2 (i.e., the risk per unit dose is reduced by a factor of 2 for low-level radiation exposures). For breast cancer, EPA assigned a DDREF of 1. Before 1993, EPA assigned a DDREF of 1 for all cancers. EPA also revised the risk estimates for leukemia based on new epidemiological data. The agency obtained the life table data from the 1979-1981 census data for the U.S. population. In the previous methodology, EPA used the U.S. population life table data during the years 1969-1971. Using the revised methodology, the cancer risk estimate has increased. For example, for low-linear energy transfer (LET) radiation (see Section 6.5.1) the lifetime fatal risk estimate associated with uniform whole-body irradiation of the U.S. population has increased by 24%, from 3.92×10^{-4}/rem** to 5.09×10^{-4}/rem for fatal cancer risk. This corresponds to an incidence risk estimate of 7.61×10^{-4}/rem. Although the results from the revised methodology were not included in the 1994 HEAST document, it is probable that the results will replace the current values in the near future (USEPA, 1994b).

As for the nonstochastic (noncarcinogenic) radiation effects, very little quantitative data, particularly at low-dose exposures, are available on mutagenesis. The majority of the evidence supporting the mutagenic character of ionizing radiation comes from extensive studies of animals. Mutation rates calculated from these studies have been extrapolated to humans and form the basis for estimating the genetic impact of ionizing radiation on humans. The teratogenic effects of radiation are better known because the fetus is much more sensitive to radiation than the mother. However, the age of the fetus at the time of exposure is the most important factor in determining the extent and type of damage from radiation exposure. Malformations produced in the irradiated embryo depend on which cells, tissues, or organs were most actively differentiating at the time of exposure. Embryos are most sensitive just after implantation until approximately eight weeks into the term; the greatest risk microcephaly (brain damage resulting in mental retardation), occurs from radiation exposures at 8 to 15 weeks.

**REM stands for Roentgen Equivalent Man and is defined as the unit of dose equivalent. The dose equivalent in "rem" is numerically equal to the absorbed dose in "rad" multiplied by the "Quality Factor" which is an LET dependent factor by which absorbed doses are multiplied to obtain a quantity which corresponds more closely to the degree of biological effect produced by x-rays or low-energy gamma rays. More simply, the rem is a measure of equivalence for the relative biological effect of radiations of different types and energies on man.

Compared to chemicals, the dose-response assessment of radionuclide is straightforward; that is, the type of effects, the probability, and the likelihood of any one of several possible adverse health effects occurring depends on and increases with the radiation dose. The severity of the effect is, however, independent of dose. Estimates of human health effects are based primarily on single, usually high (acute) doses of radiation. To describe these effects as a function of dose, assessors use the "linear model." That model assumes that there is no threshold for the induction of cancer or genetic effect (a conservative assumption and model). There are very few data on the effects of radiation at low doses or the effects of chronic, long-term exposure in humans.

6.4.2 Radiation Health Effects in Humans

Four major, scientifically august bodies have been responsible for collecting and evaluating data on the human health effects of ionizing radiation: the Committee on the Biological Effects of Ionizing Radiation (BEIR) of the National Research Council/National Academy of Science, the National Council on Radiation Protection and Measurements (NCRP), the International Commission on Radiological Protection (ICRP), and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). In the United States, EPA is responsible for developing guidelines for radiation risk assessment and has relied on the published evaluations of these groups.

6.5 Risk

6.5.1 Cancer Risks

For radiation risk evaluation, the "type" of radiation is usually separated into either low or high Linear Energy Transfer (LET) effects: LET refers to the rate at which energy is deposited as the particle or gamma ray travels through matter, Radionuclide that emit radiation as alpha particles or neutrons are classed in the high LET radiation category; beta particles, x-rays, and gamma rays are considered low LET radiation. Some radionuclides emit both types of radiation (e.g., radium-226 emits both high LET alpha and low LET gamma radiation). By far the more dangerous more cytotoxic, and more carcinogenic of the two is high LET radiation. However, that does not mean that low LET is not also dangerous, cytotoxic, and carcinogenic. For fatal cancers, the lifetime exposure period has a risk factor of 5.09×10^{-4}/rad for low LET radiation and 3.1×10^{-3}/rad for high LET. The corresponding incidence risk factors for all radiation-induced cancers (lifetime exposure) is 7.6×10^{-4}/rad for low LET and 5.0×10^{-3}/rad for high LET (USEPA, 1994b).

6.5.2 Genetic Risks

The genetic effects of ionizing radiation have been studied extensively in plants, animals, and various human populations such as the Japanese atomic-bomb survivors. Results of the A-bomb studies have consistently yielded estimates of genetic effects that imply lower risks in humans than in animals. This indicates that humans are less sensitive to radiation induction of mutations in germ cells and that the risks derived from animal data will be conservative in humans. As a result of this, BEIR V (NAS, 1990) and UNSCEAR (UNSCEAR, 1988) have based their genetic risk estimates on the lower 95 percent

confidence limit for the Japanese data, which is consistent with the mutation rate doubling doses found in mice (doubling dose is the dose that doubles the incidence of genetic defects).

The doubling dose for humans is estimated to be 100 rem; the expected genetic effects per rem per 30-year generation (the generally accepted reproductive span) are less than one to about 100 per million liveborn offspring for multi-generations. The natural incidence for genetic anomalies from all causes ranges from 400 to 30,000 per million liveborn offspring. EPA currently estimates that exposure to 1 rad per generation of lowdose-rate, low-LET radiation will induce 260 cases of serious heritable disorders per 10^6 live births in all generations. For high-LET radiation, the estimate is 690 serious heritable disorders per 10^6 live births in all generations. These risks are based on BEIR III (NAS, 1980), which are essentially the same as in BEIR V (NAS, 1990). The main difference lies within the reassessment of doses assigned to the A-bomb survivors, the effect of which, in general, will increase the risk of low-LET radiation calculated according to a particular model. Attempts to estimate doubling doses from data on Japanese atomic-bomb survivors have consistently led to values larger than those derived from the animal data, and, consequently, they imply lower risks. Although risks calculated from animal data have large confidence intervals, estimates from those exposed to radiation in Hiroshima and Nagasaki are known with even less precision. In spite of these uncertainties, the data suggest a real difference, with the estimated lower 95% confidence limit of the human data approximating the median of a large number of values obtained in mice. If it is assumed that the apparent difference is real, humans would be less sensitive to radiation induction of mutations in germ cells than mice, and the risks in BEIR III should be considered more conservative. The BEIR V Committee stated that despite all the careful work that has gone into their collection and analysis of the human data, they are in no better position to decide the issue than the previous committees (BEIR I, HI, & IV. Note: The BIER II report was strictly a cost/benefit analysis.)

6.5.3 Developmental Risks

Developmental or teratogenic effects are somatic effects resulting from exposure of the unborn, in-utero, to ionizing radiation. These effects differ from genetic effects in that they cannot be passed on to other generations but can include severe mental retardation, microcephaly, and other structural abnormalities. The extrapolation of animal data to humans is extremely difficult because of the significant differences in fetal development rates. EPA's current estimate is 4,000 effects per rad during 8 to 15 weeks of gestation for low LET radiation (mainly x- or gamma ray radiation) (USEPA, 1994b).

6.5.4 Radiation Risk Calculation Methodology

The methodology for estimating the total lifetime excess cancer risks due to continuous, lifetime exposure, based on ICRP 23's recommended 70-year life span, is broken down into risk characterization for internal (inhalation or ingestion) and external (mainly gamma emitters) exposures.

Internal Exposure

The internal risk characterization is calculated from:

$$Risk = I \times SF$$

Where:

Risk = cancer incidence, expressed as a unitless probability

I = lifetime radionuclide intake (pCi)

SF = slope factor $(pCi)^{-1}$

The slope factor is either a HEAST value for a particular radionuclide or the sum of the HEAST slope factors for that radionuclide and its short-lived progeny to account for ingrowth (USEPA, 1989a; USEPA, 1989b).

External Exposure

External risk characterization for gamma-emitting radionuclide in surface soil (photon-emitting radionuclide distributed uniformly in a thick layer of soil, expressed as risk/yr per pCi/gram of soil) is calculated from:

$$Risk = (SF)[(C_s)(EF)(ED)(ET)(1\text{-}SH)]$$

Where:

Risk = risk of cancer incidence, expressed as a unitless probability

SF = radionuclide slope factor, (risk/yr/pCi/g) from EPA HEAST tables

C_s = radionuclide soil concentration (pCi/g)

EF = modifying factor, fraction of year exposed (unitless)

ED = exposure duration (years)

ET = fraction of&y exposed (unitless)

SH = shielding factor (titles)

External slope factors do not include the radiation contributions from radioactive decay progeny. In order to include this additional, and often substantial, radiation in the overall risk calculations, a computer program (Rad Decay) must be used to predict future radiation levels from progeny when parent isotopes are decayed over a period of time.

The calculation of a risk characterization for gamma emitters from sources other than surface soil is

$$Risk = (DE)(RC)$$

Where:

Risk = risk of cancer incidence (titles probability)

DE = total dose equivalent (rem)

RC = cancer risk coefficient (rem-l)

This deviation is necessary because the EPA slope factor method is not applicable to gamma-ray exposures from sources other than contaminated surface soils. The cancer risk coefficient is not radionuclide-specific. Consequently, the same coefficient is used in all cases to which this method applies.

6.6 Radiation Units Conversion

6.6.1 SI Units

The transition to the International System of Units (SI) has been recommended by numerous international technical and regulatory organizations and it is the policy of the United States that regulations should not impede this transition. To ensure a smooth transition to SI units, international and domestic organizations have recognized that there are circumstances where there is a need for showing information in both the SI and the customary units (NAS, 1990).

The Curie and Becquerel are units of measure of the quantity or activity of radioactive material which indicates the rate that atoms in the material are giving off radiation or disintegrating. The Curie (Ci) is equal to 37 billion disintegrations per second whereas the Becquerel (Bq) is equal to only one disintegration per second. The sievert (Sv) and the rem are units of measuring absorbed energy from all types of radiation in human tissue (dose equivalent health effects). The Gray (Gy) and rad are the units of measuring the absorbed dose of radiation energy in matter.

6.6.2 Conversion Factors

- Becquerel (Bq) = 1 disintegration per second = 2.7×10^{-11} Ci
  Curie (Ci) = 3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq

- Gray (Gy) = 1 joule per kilogram = 100 rad
  Rad = 100 erg per gram = 0.01 Gy
  Rem = 0.01 Sievert (Sv)

- Sievert (Sv) = 100 rem

For radon and short-lived radon daughters only:

Working Level (WL) = 1.3 x 10^5 MeV of alpha energy in 1 liter of air (approximately = 100 pCi of radon-222 alpha energy released from decay daughters)

Working Level Month (WLM) = Exposure from 1 WL of radon daughters for 170 working hours

6.7 Regulator Dialogue

The statutory and regulatory history shows that radionuclide effects are to be considered in the baseline risk assessment process. The guidance documents reveal that risk from radionuclide and other chemicals are calculated differently. An expert in radiation risk should be consulted to ensure that the risks calculated for the radionuclides have been estimated properly. Mortality-to-incidence risk ratio data is included in cancer risk calculations for radionuclide. Radioactive decay progeny must be considered for each radionuclide at the site.

Gamma emitters must be considered for internal and external exposures. Gamma radiation is also an externally penetrating exposure. Alpha and beta emitters should only be considered for internal exposures. The exception to this is if the alpha or beta emitter is associated or attached to a dermally absorbed chemical.

6.8 References

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